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Inhibition of TGF-β-induced Collagen Production in Rat Achilles Tendons by Injection of Tetramethylpyrazine

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1 Introduction

Postoperative adhesions frequently compromise the success of tendon repair. Manipulation of growth factors responsible for scar formation may decrease adhesion formation. Transforming growth factor β (TGF-β) is a key cytokine in the pathogenesis of tissue fibrosis and tendon adhesions. Among kinds of agents that have been used to modulate the process of tendon adhesion formation, tetramethylpyrazine, an alkaloid extracted from a Chinese herb, Ligusticum Chuangxiong, has been shown to reduce adhesion in healing tendon in both experimental animal models and clinical studies. The purpose of this study is to examine the effectiveness of tetramethylpyrazine in blocking TGF-β-induced collagen I production in rabbit flexor tendons in vivo.

2 Materials and Methods

Injection of tetramethylpyrazine was performed into the Achilles tendon sheath of 20 rats right after tendon transection operation. 20 control rats also had tendon transection without injection of tetramethylpyrazine. Tendon samples, around the original transection sites, were harvested at 7^{th} day after surgery. Collagen type I production was measured by enzyme-linked immunoabsorbent assay and TGF-β bioactivity was measured by the luciferase assay. Results from experimental groups were compared with controls.

3 Results

The addition of tetramethylpyrazine significantly reduced TGF-β-induced collagen type I production ($p < 0.05$). TGF-β bioactivity was also reduced by tetramethylpyrazine ($p < 0.05$).

4 Discussion

This study showed that TGF-β inhibition through tetramethylpyrazine was effective in healing tendon cells. Yet, similar studies showed that VEGF, another growth factor that is involved in scar and adhesion formation was also reduced by tetramethylpyrazine during tendon healing. The molecular mechanisms of tendon scar formation decrease by tetramethylpyrazine remain unclear, however. These results encourage further experiments that use the agent to modulate tendon wound healing in the hope of eventually revealing the mechanisms of tendon adhesion.

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